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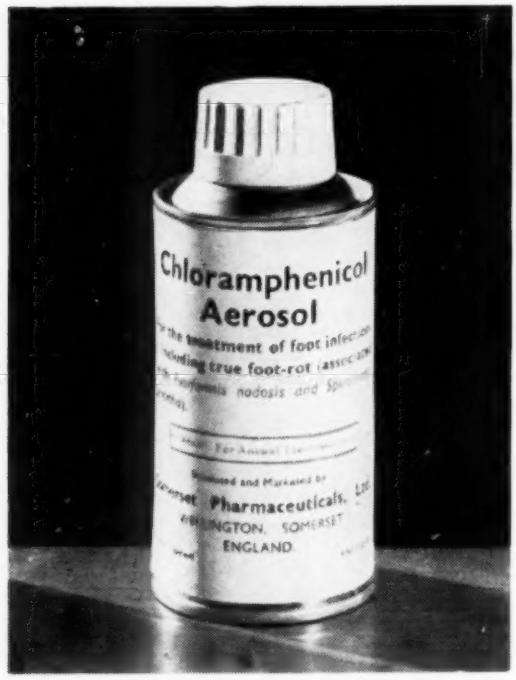

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Pulmonary Aspergillosis in Lambs

BY

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AND

C. V. WATKINS

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SUMMARY.—7 cases of pulmonary aspergillosis in lambs, one of them subclinical, are described.

A. fumigatus *was recovered from all lung lesions. Three types of hyphal growth were observed in the lesions.*

PULMONARY aspergillosis is world-wide in distribution and its occurrence has been reported in many domestic animals and birds as well as in wild species (Ainsworth & Austwick, 1959). So far as we are aware there are only 4 reports of this condition in sheep: by Mazzanti in Italy (1891), van Hellens in Finland (1902), Andersen in Denmark (1927) and in the Annual Report of the New Zealand Department of Agriculture (1955). There is no published record of the occurrence of the disease in Great Britain although one of us (C.V.W.) some years ago encountered cases of severe pulmonary congestion and oedema in a flock of ewes which had been fed on some extremely mouldy roots and it seems likely that this was the earliest observation of this type of disease in this country. The purpose of this paper is to describe 7 cases of pulmonary aspergillosis encountered by us in sheep in south and south-west England.

Case Reports

No. 1—*February* 25*th*, 1955. *Cornwall*

A three-week-old lamb unsuccessfully treated with streptomycin in an outbreak of joint-ill associated with Streptococcus group C infection.

Post-mortem findings: extensive congestion of both lungs; scattered throughout the lungs were numerous greyish-white nodules, 1 to 3 mm. in diameter each surrounded by a narrow haemorrhagic zone.

Nos. 2 *and* 3—*February* 28*th*, 1955. *Devon*

Three-week-old lambs from a flock in which there were unexplained losses.

Post-mortem findings: *No.* 2 severe pulmonary congestion with a small number of nodules similar to those in No. 1 present in all lobes. *No.* 3 severe pulmonary congestion without nodular lesions.

No. 4—*November* 8*th*, 1957. *Surrey*

An eleven-day-old Dorset Horn orphan ram lamb, bottle fed by the shepherd, died after gradual loss of condition.

Post-mortem findings: a debilitated carcase; pulmonary lesions as in case No. 1 (Fig. 1.); the bronchial and mediastinal lymph nodes congested; urinary bladder distended; bilateral hydronephrosis and partial occlusion of the urethra by cellular debris at the sigmoid flexure.

No. 5—*April* 30*th*, 1958. *Surrey*

A six-day-old Dorset Horn ewe lamb, born as twin to a mummified foetus, very weak from time of birth and deteriorating gradually in condition. When 1½ days old was too weak to suck and was brought with the ewe into a straw-bedded shed where it was bottle-fed on cow's milk 4 times daily.

Post-mortem findings: severe pulmonary lesions similar in character and distribution to No. 4 but the nodules smaller in diameter (0.5 to 1.5 mm.). No other abnormality.

No. 6—*May* 1*st*, 1958. *Surrey*

A seven-day-old Dorset Horn ewe lamb born on the same day and in the same field as No. 5 and also a twin to a mummified foetus. Although it was a strong looking lamb and sucked well, it was brought with its dam on the same day into the shed where lamb No. 5 and its ewe were kept. It was an apparently normal lamb but as it had been exposed to the same environmental conditions as lamb No. 5, it

was destroyed the day after the latter died, in the hope that it would provide information on individual susceptibility to *Aspergillus fumigatus*.

Post-mortem findings: numerous greyish-white nodules 0.4 to 0.8 mm. diameter present in both lungs, which were otherwise normal in appearance; no other lesions were noted.

No. 7—October 27th, 1958. Surrey

A ten-day-old Dorset Horn ram lamb. The flock was kept in a field where the animals had access to bales of mouldy straw. Several lambs were affected with joint-ill and were responding to penicillin treatment but 2 apparently healthy lambs were unexpectedly found dead. One was submitted for laboratory examination with a tentative diagnosis of lamb dysentery.

Post-mortem findings: almost complete consolidation of both lungs with very numerous lesions similar to those in Nos. 1, 2, 4 and 5 (Fig. 2).

Mycological and Bacteriological Examination

All carcases were subjected to routine bacteriological examination but no known pathogens were recovered; the media used were 5 per cent. sheep blood and MacConkey agar incubated aerobically at 37° C. No evidence of *Cl. welchii* toxins was found on biological examination of the intestinal contents of lamb No. 7.

Direct microscopical examination of the lung lesions was carried out by shelling out the nodules, mounting them in water or lactophenol, teasing with needles and crushing lightly under a coverslip. Numerous Aspergillus-type hyphae were observed, varying in morphology from short, swollen, frequently septate filaments to narrow, elongated, branching hyphae. In some cases the swollen hyphae were surrounded by sheaths of refractive material bearing short projections.

A. fumigatus was recovered from the lung lesions of all lambs by both direct and dilution plating of teased parts of the nodules, using 2 per cent. malt agar containing 20 units penicillin and 40 units streptomycin per ml., incubated for 24 hours at 37°C.

Histological Examinations

Tissues were fixed in 10 per cent. formal saline and sections stained with haematoxylin and eosin and the periodic acid-Schiff and Gomori-Gridley stains.

Various stages of purulent nodular pneumonitis were observed. The early lesions were present in the alveoli, often close to small bronchi or bronchioles, and consisted of a central fungal colony up to 100μ in diameter composed of short-celled, swollen and branching hyphae 5.0μ to 12.0μ diameter, surrounded by polymorphonuclear cells, cellular debris and macrophages (Fig. 8). Compression of the alveoli at the periphery of the nodules was very marked. The small bronchi and bronchioles were frequently plugged with purulent exudate, the blood vessels were generally dilated and congested and some, occluded by thrombi, were invaded by hyphae. In older lesions, as seen in lamb No. 7, macrophages and epithelioid cells predominated with fibroblasts appearing on the periphery of the nodules (Fig. 5). The hyphae in the centre of these lesions were swollen and short-celled but had given rise to narrow apical branches which divided frequently at the periphery of the lesion, forming the so-called "actinomycetoid" form (Fig. 3). The central swollen hyphae of these older lesions were usually ensheathed in eosinophilic material 1.5μ thick bearing blunt finger-like projections 3.0μ to 4.0μ long, these structures being referred to as "asteroids" in other mycoses, *e.g.* coccidioidomycosis. In No. 6 all the lesions were very small and undergoing resolution. Many of the hyphae were disintegrating, and those intact mostly had asteroid sheaths (Fig. 4).

A marked variation in the staining properties of the hyphae was seen in the different stages of growth. All the swollen hyphae in the centres of the lesions stained deeply with the periodic acid-Schiff and Gomori-Gridley techniques but failed to stain with either haematoxylin or eosin. On the other hand, the "actinomycetoid" type of growth on the periphery of the lesions was basophilic and stained well with the haematoxylin and eosin technique but not by the other two methods.

Discussion

Little is known about the clinical manifestation of the disease and we have been unable to confirm the reports of coughing made by van Hellens (1903) and Andersen (1927). The former described an outbreak of pulmonary aspergillosis in a flock of adult sheep affected with a dry cough and progressive emaciation, whilst the latter encountered the disease in 2 weak and undersized lambs which had come from a flock in which severe coughing had occurred. The striking feature of the outbreak recorded some years ago by one of us (C.V.W.) was sudden death and evidence of inhalation of such a mass of spores that the mucous membrane of all air passages was covered with the typical green colour of *A. fumigatus* spores. No evidence of nodular lesions was found in the lungs.

The 7 cases described in this paper showed various stages from the acute to the early chronic but we were unable to demonstrate giant cells as described by van Hellens (1903).

Three types of hyphal growth could be distinguished and appeared to be related to the stages of development of the lesions. The swollen hyphal cells and inflammatory exudate in the alveoli may be regarded as typical of the acute stage of the disease as observed in lambs 1, 2, 4, 5 and 7, whilst the closely branched actinomycetoid type of hyphal growth at the periphery of the necrotic areas in most of the lesions in No. 7 and in one lesion in No. 1 is thought to appear at the beginning of the chronic state which was described by van Hellens (1903). The third type of hyphal growth was seen in lambs 2 and 3 in the hyperaemic areas away from any of the nodular lesions and this closely resembled the rapid vegetative growth produced in submerged culture (Fig. 7).

Whether the presence of lesions of different ages in the same tissue indicates continuous infection or spread from lesions already present could not be ascertained, although this point is of considerable importance in a study of the pathogenesis of the

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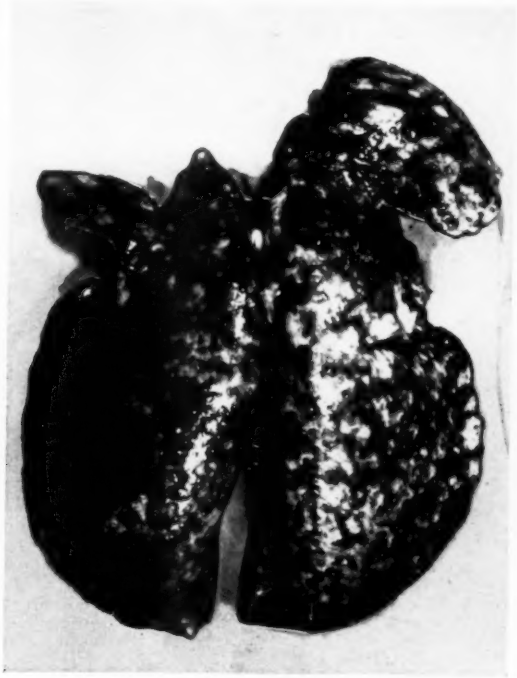

FIG. 1.—Lungs of 11-day-old lamb. Case No. 4.

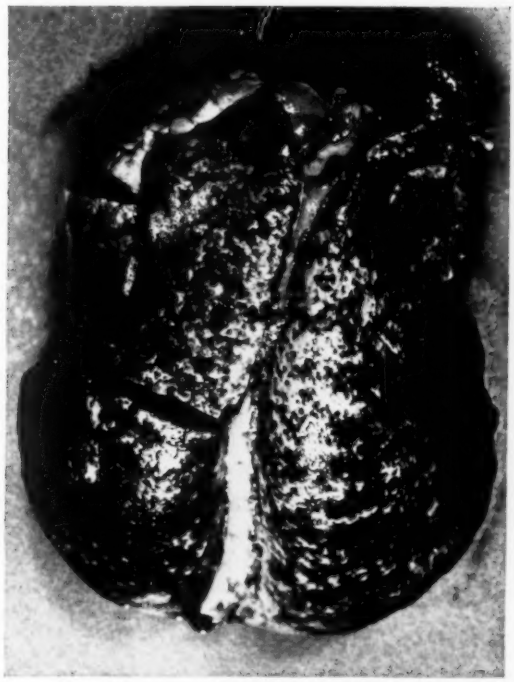

FIG. 2.—Lungs of 10-day-old lamb. Case No. 7.

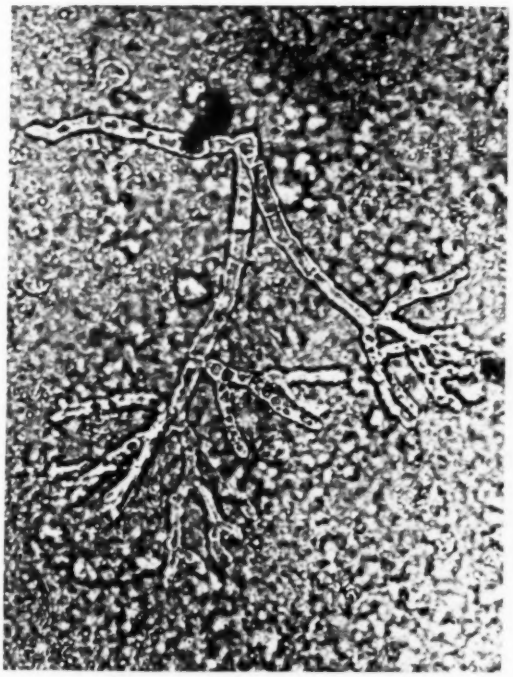

FIG. 3.—Hyphae of *A. fumigatus* showing early development of actinomycetoid type of branching. Unstained fresh mount. Case No. 5. × 900.

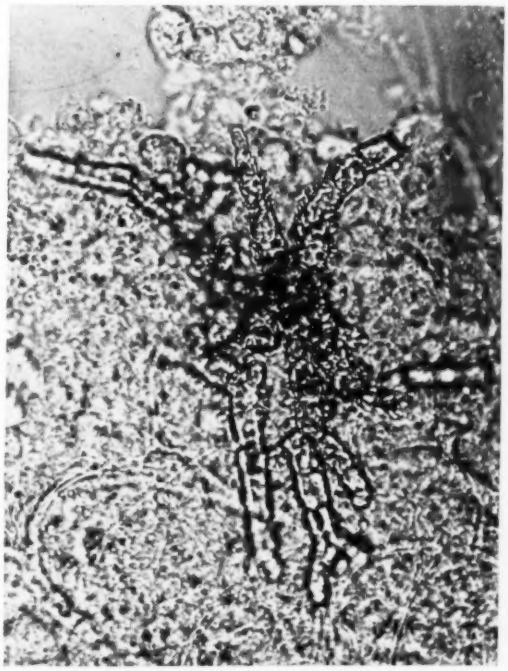

FIG. 4.—" Asteroid " sheath around hyphae in an early lesion. The hyphae tips have no sheath. Unstained fresh mount. Case No. 6. × 900.

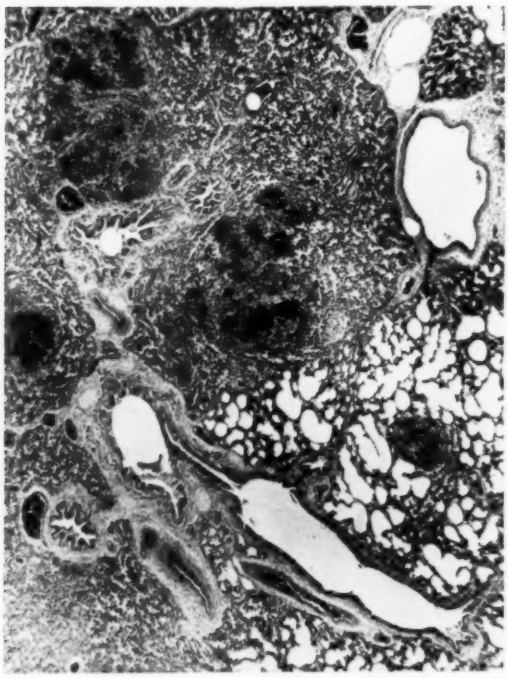

FIG. 5.—Section of lung in Case No. 5. Stained H. and E. × 50.

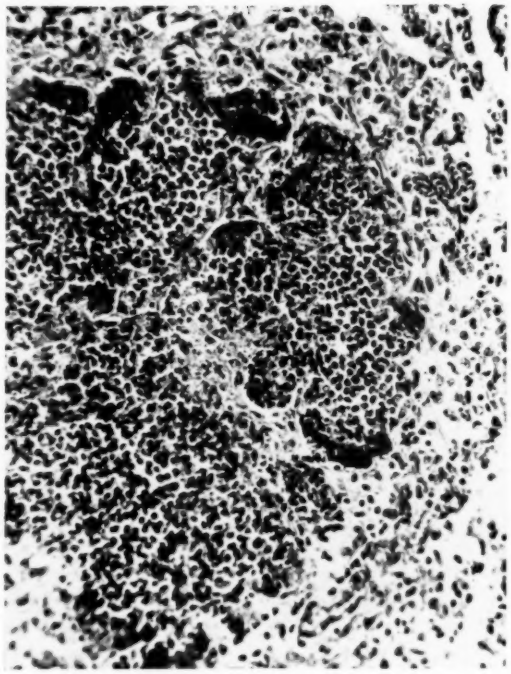

FIG. 6.—Cellular infiltration of alveoli in Case No. 4. Stained H. and E. × 200.

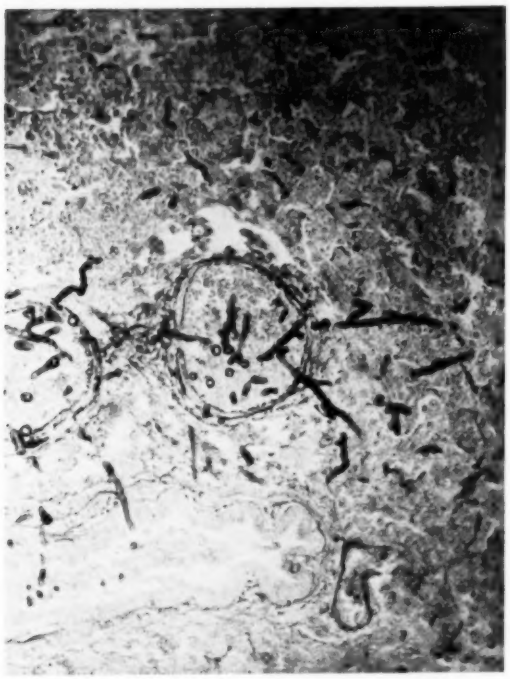

FIG. 7.—*A. fumigatus* hyphae in free-growing invasive stage. Case No. 2. Stained Gomori-Gridley. × 300.

FIG. 8.—Swollen hyphae of *A. fumigatus* in centre of a lesion. Case No. 1. Stained Gomori-Gridley. × 900

Field Trials with Trolene* (Dow ET-57, Ronnel, Etrolene) against Warble Fly Grubs in Cattle

BY

W. N. BEESLEY

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SUMMARY.—This paper compares two seasons' use of "Trolene" against warble flies in Britain. The drug appeared rather more satisfactory in 1957-58 *than in* 1958-59.

Pulmonary Aspergillosis in Lambs—*Concluded.*

disease. Detection of a subclinical case in lamb No. 6 is of interest and may indicate that healthy lambs can overcome the infection. This may account for Andersen's (1927) failure to reproduce the disease experimentally in healthy lambs in which he found no lesions when they were killed 2 months later. Had lamb No. 6 been destroyed at a later date probably no lesions would have been found on *post-mortem* examination.

In spite of the very few reported cases of ovine pulmonary aspergillosis, it is possible that the condition is far more common than is generally realised. The close resemblance of the lesions to those of *Muellerius capillaris* already commented upon by Andersen (1927), and the possibility of subclinical infection could easily result in cases of aspergillosis not being identified. When a larger number of cases has been recorded, it should be possible to distinguish the different clinical manifestations with reference to the type of lesions, individual susceptibility and the effect of environmental factors which in our opinion exert a very strong influence on the course of the disease.

Acknowledgments.—We are indebted to Mrs. J. C. Cartner for technical assistance and to Mr. E. Clements for the photographs.

Postscript

Since this paper was prepared 4 further cases of aspergillosis in lambs have been reported, one each in Devon and Cornwall (C.V.W.), an outbreak in Cambridgeshire by J. B. Spence (1959) and a single case in Staffordshire by H. E. Roberts (1959).

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DURING the last 5 years much progress has been made in the development of organo-phosphorus systemic insecticides for killing warble fly larvae, *Hypoderma lineatum* and *H. bovis*, during their migration through the body of the host. Trolene* (Dow ET-57, Ronnel, Etrolene) shows considerable promise and work has been carried out on this compound in America (McGregor & Bushland, 1957; Jones, 1959), England (Kendall, 1958; Harrison, 1958) and Ireland (Hatch, 1958; Kenny & Thornberry, 1958, 1958). The Ministry of Agriculture arranged a successful preliminary trial of Trolene in 1957-8 and the results were briefly reported by Kendall (1958). The present paper records these results in more detail and compares them with those of a second trial conducted in 1958-9.

First Trial 1957-8

Three of the Ministry's Veterinary Investigation Centres co-operated: Aberystwyth, Reading and Starcross (Exeter). Ninety-four cattle were treated, at the rate of 110 mg. Trolene per kg. bodyweight, and 100 kept as controls. All cattle were treated either in November or January.

The results of this trial appear in Table I.

Second Trial 1958-9

Four Veterinary Investigation Centres participated: Aberystwyth, Reading, Starcross and Bristol. Two hundred and ninety-seven cattle were treated, at the same dosage rate as before, and a further 167 used as controls.

This trial was designed to show any difference in the degree of warble control following different times of treatment. Each group of treated animals was dosed once, but dosing was spread to cover the period from September to January inclusive. Boluses and wettable powder were used in the 1957-8 trial, and mainly wettable powder in the second trial. Both Trolene formulations contained 40 per cent. w/w of the active ingredient. Some cattle developed toxic symptoms of the type described by Harrison (1958), but there were no deaths. Young animals were selected for these experiments as it is generally accepted that they are more likely to be heavily infested with warbles than older cattle. None of the animals used in the trials was more than 2½ years old at the time of dosing.

The results from this trial appear in Table II.

* Trolene = 0, 0 – dimethyl 0 – 2, 4, 5 – trichlorophenyl phosphorothioate.

TABLE I
RESULTS OF THE FIRST FIELD TRIAL, 1957–8

Location	Dosed	No.	Examinations, warbles per head		
1. *Starcross*			Feb., 1958	Mar., 1958	Apr., 1958
Farm A	Jan., 1958	20	0·45	0·9	—
	Controls	16	13·1	26·1	—
	% control	—	97	97	—
Farm B	Nov., 1957	19	0	0·05	0·3
	Jan., 1958	7	0	0	0
	Controls	26	1·7	6·2	9·0
	% control	—	100	99–10	97–100
2. *Aberystwyth*			Feb., 1958	Apr., 1958	May, 1958
Farm A	Nov., 1957	20	0·85	1·25	0·55
	Controls	28	6·2	14·5	7·0
	% control	—	86	91	92
3. *Reading*			Mar., 1958	Apr., 1958	May, 1958
Farm A	Jan., 1958	10	0·1	0·2	0
	Controls	11	3·1	9·2	9·0
	% control	—	97	98	100
Farm B	Jan., 1958	10	0	0	0
	Controls	9	6·3	13·0	6·6
	% control	—	100	100	100
Farm C	Jan., 1958	8	0	0	—
	Controls	8	7·8	10·9	—
	% control	—	100	100	—

Where a range of percentage control is indicated, the figures refer to control from treatment on two different dates.

TABLE II
RESULTS OF THE SECOND FIELD TRIAL, 1958–9

Location	Dosed	No.	Examinations, warbles per head		
1. *Starcross*			April, 1959		
Farm A	Oct., 1958	31	6·8 (83% control)		
	Dec., 1958	30	3·5 (91% control)		
	Controls	15	40·5		
			April, 1959		
Farm B	Sept., 1958	11	0·1 (98·5% control)		
	Nov., 1958	10	0·0 (100% control)		
	Feb., 1959	10	0·1 (98·5% control)		
	Controls	19	7·0		
2. *Aberystwyth*			Feb., 1959	Mar., 1959	Apr., 1959
Farm A	Nov., 1958	37	1·2	1·7	4·1
	Controls	36	2·3	4·8	9·7
	% control	—	48	65	58
Farm B	Jan., 1959	28	0	0·3	0·4
	Controls	27	0·9	2·3	5·0
	% control	—	100	87	92
3. *Reading*			Mar., 1959	Apr., 1959	May, 1959
Farm A	Oct., 1958	8	1·7	3·4	2·4
	Jan., 1959	8	0·7	1·0	0·5
	Controls	6	0·9	2·0	2·9
	% control	—	0–22	0–50	17–83
Farm B	Oct., 1958	32	4·5	9·6	—
	Jan., 1959	32	1·2	0·2	—
	Controls	15	11·1	16·2	—
	% control	—	59–89	41–99	—
4. *Bristol*			Mar., 1959	Apr., 1959	May, 1959
Farm A	Oct., 1958	42	2·7	7·5	7·8
	Jan., 1959	48	1·6	1·7	2·7
	Controls	47	11·6	17·8	14·2
	% control	—	77–86	58–90	45–8.
Farm B	Feb., 1959	28	1·1	2·1	4·1
	Controls	27	3·3	5·9	7·9
	% control	—	67	64	48

Discussion

In the first trial, control varied from 86 per cent. upwards. Usually it was at least 90 per cent. In the second trial there was no control in certain groups of cattle at some examinations for warbles and percentage control was usually much less than 90 per cent. The Starcross results were similar in both trials, but the Aberystwyth and Reading results did not appear as satisfactory in 1958-9. Only moderate control was noted in the Bristol experiment (1958-9 only).

The apparently less spectacular results in the second trial are due, to some extent, to the lower incidence of infestation appearing in the untreated control animals. At Reading (Farm A, 1958-9) untreated cattle in one group had fewer warbles than treated cattle at 2 of the 3 examinations.

1958 was very wet in comparison with the previous 25 years (Smith, 1958) and this might be expected to have affected the flight and egg laying activity of the female warble flies, and perhaps also the entrance of newly-hatched larvae into the host, leading to an over-all lower incidence of infection in cattle in 1959. Such a view is confirmed to some extent by independent observations made from this laboratory on the incidence of *H. lineatum* in the gullets of slaughtered cattle. A much lower percentage of infection was noted in 1958-9 compared with 1957-8 (Beesley, unpublished data).

While this may in part explain the apparently less satisfactory results noted in 1958-9 it is plain also that variable results following the use of Trolene have been noted by some other workers. In Ireland, Hatch (1958) recorded 93 and 97 per cent. control but Kenny and Thornberry (1958) had only 52, 57, 71 and 80 per cent. control. In further trials (Kenny and Thornberry, 1959) control varied from 88 to 98 per cent. In America, most trials with Trolene have apparently given excellent results, *e.g.* DeFoliart (1958) had 88 to 98 per cent. control with doses of 85 to 110 mg. per kg. and Wade and Colby (1958) reported 84 to 98.5 per cent. control in a series of experiments, although another trial gave only 45.8 per cent. control.

Little difference between time of treatment and percentage control was observed in the present trials and this agrees with the results of Harris *et al.* (1959) in America.

Both *Hypoderma lineatum* and *H. bovis* were present in the areas used for the Ministry trials, but far more *H. bovis* than *H. lineatum* larvae were submitted for examination. *H. bovis* is believed to be the more common species, and emerges later in the year. Most of the grubs received at the laboratory

(*Continued at foot of col. 1 overleaf*)

A Case of Hydroquinone Developer Poisoning in a Boxer Puppy

BY

TREVOR TURNER

Northolt, Greenford, Middlesex

SUMMARY.—A case of hydroquinone developer poisoning in a boxer puppy is described which showed several unusual features. A survey of the literature revealed no standard treatment was available, therefore it was considered that the following notes might be of value.

Field Trials with Trolene—*Concluded.*

were sent towards the end of the warble fly season, in May and June. It is just possible that there was a real difference in the proportion of the 2 species in 1959 compared with 1958, this leading to a reduced degree of control by differential effect of the insecticide. So far, however, there is no experimental evidence to suggest that Trolene has a different effect on the 2 species of *Hypoderma*.

Trolene has given good results in many experiments, both in America and in Britain, but is plainly not a complete answer to the warble problem. More information is still needed on the mode of action of this type of compound on migrating warble larvae in order to find the most effective use under field conditions. Until more complete data are available there seems every reason to treat cattle at as early a stage in the infection as possible, both to minimise damage from the activity of the parasite and to avoid the possibility of side effects resulting from the death of larger parasites either in the spinal canal or elsewhere.

Acknowledgments.—I wish to thank Messrs I. H. Fincham, L. E. Hughes, R. M. Loosmore and J. A. J. Venn, of the Ministry staff, for their co-operation in arranging trials at their respective Veterinary Investigation Centres.

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History

ON July 8th, 1959, a 5-month-old brindle boxer bitch was presented at the surgery as an emergency case, with a history of having eaten a quantity of developer. The owner had in his possession several packets of home developer which the dog had chewed. Upon discovery, one of these packs, which had been produced in South America and contained elon, sodium sulphite, hydroquinone, sodium carbonate and potassium bromide, was found to have been badly chewed. The owner was not unduly worried until half an hour later when it was noticed that the bitch was breathing heavily and vomiting violently.

Examination

The animal had been walked approximately ½ mile in hot sunlight. Upon examination the bitch was noticed to be in a state resembling surgical shock ; she was breathing fast and shallowly and her membranes were very pale with the exception of the conjunctival membranes which were very congested. During the examination she became incoordinate, trembling violently and finally trying to vomit without result. Her pulse was 180, but her temperature was normal.

The owner had brought with him the remains of the developer pack, examination of which showed that it was mainly the hydroquinone that was missing.

Prognosis

A guarded prognosis was given and the bitch was retained at the surgery. An emetic of sodium carbonate (washing soda) was administered while a resort was made to the literature. Examination 15 minutes later showed that the bitch was now evincing signs of photosensitisation similar to that seen by the author in cattle. Those parts of the animal which had been exposed to the sunlight were swelling rapidly. The conjunctival membranes were now more congested and the animal's head swollen up to a degree suggestive of anaphylaxis.

Treatment

A search of the literature proved of no avail and contact was made with several children's hospitals in the hope that they might be able to help with an antidote. In the meantime, an intramuscular injection of 50 mg. promethazine hydrochloride* was administered.

Although several developer substances are recorded as having been swallowed by children, there was apparently no knowledge of the substance involved in this particular case. Finally, the Forensic

* Phenergan : May & Baker, Ltd.

Laboratory, New Scotland Yard, was contacted and it suggested the administration of large quantities of milk and castor oil, plus the universal antidote which consists of: magnesium oxide—one part; tannic acid—one part; activated medicinal charcoal—two parts. This mixture in 4 oz. of water was administered together with ½ oz. of castor oil (Lucas, 1953). Four hours later the bitch appeared somewhat better and her pulse rate was 120. The mucous membranes had returned to a normal colour. The conjunctival membranes were now very ulcerated and hydrocortisone and neomycin* ointment was applied. Twelve hours after administration the bitch collapsed. The condition of photosensitisation returned. A blood transfusion was given, using one pint of venous blood from a 7-year-old pointer bitch which, on a rapid plate test, appeared to be compatible. The blood was injected slowly into the radial vein. A further 50 mg. of promethazine hydrochloride solution was administered intramuscularly.

Over the next 8 hours the bitch improved steadily, although she vomited approximately every 2 hours. She was encouraged to take quantities of sweetened warm milk. Areas around the eyes and mouth—areas of swelling which had been noticed previously—ulcerated. The bitch was slightly "photophobic."

Twelve hours after the transfusion (approximately 24 hours after ingestion) she became very excitable, almost to the point of having a fit and 25 mg. chlorpromazine hydrochloride† was administered intramuscularly.

Eighteen hours after the transfusion (approximately 30 hours after ingestion) she had ceased to vomit and was able to take nourishment in the form of egg and milk. She subsequently became quite lively and to all intents and purposes normal, except for ulceration of the previously oedematous areas over the face and head. These were further treated with penicillin‡ parenterally and neomycin and hydrocortisone ointment topically. Sollman in his Manual of Pharmacology (8th edition) 1957, 811–12 indicates that hydroquinone produces leucopaenia. Perhaps this continued ulceration could be explained by an agranulocytosis in the areas affected.

For a further period of 3 days there was some photophobia. After a period of 7 days the local lesions had healed.

Further Progress

Six weeks after discharge the bitch was returned to the surgery with an inexplicable oedema of the larynx which responded to promethazine therapy. It was noticed that the skin over the previously oedematous areas had now thickened considerably and was showing keratitis.

Sollman quotes Schwartz (1939) as having reported a change in hair colour in cats from black to grey. As a matter of interest the bitch was examined again on November 2nd, 1959, approximately 4 months after ingestion: the previously oedematous ulcerated areas could still be detected by the thickening of the skin but no change in hair colour was detected.

Conclusion

The most salient feature of this case was the apparent photosensitisation of the bitch. It would be interesting to know if this phenomenon has been observed previously following the ingestion of a similar chemical substance, since it is difficult in this case to decide whether the hydroquinone itself was responsible for the condition or whether it was merely non-specific reaction of the animal to a noxious substance.

Acknowledgments.—I am indebted to the Forensic Laboratory of New Scotland Yard for the help and interest they took in this case: also to Kodak Ltd. and the Royal Veterinary College for their help and attention.

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* Occulentum, Efcortilan and Neomycin: Glaxo Laboratories.
† Largactil: May & Baker, Ltd.
‡ Crystapen: Glaxo Laboratories.

MR. MICHAEL WHITMORE

Dr. I. Tucker Burr writes from Vermont, U.S.A.:—

I wish to report the death of Michael Mervyn Whitmore, M.R.C.V.S., in Bellows Falls, Vermont, on December 19th, 1959. Mike came to New Hampshire in February, 1958, and became my partner in 1959. In his short years here in this least cosmopolitan section of the United States his buoyant personality charmed all those who met him. His successful career was, however, overshadowed by the malignancy that had been with him and which finally overcame his courage and resolve. He leaves his wife, Joan, and two children, Jamie and William, and many friends here who will remember him.

THE STEELE-BODGER MEMORIAL PRIZE

This prize was created by the fund established in 1953 to perpetuate the memory of the late Harry Steele-Bodger. It takes the form of a travelling scholarship or a contribution to a tour of study abroad. In addition, applications for grants of small amounts to enable practising members of the profession to make short study tours abroad receive sympathetic consideration.

Those eligible are members of the Royal College of Veterinary Surgeons or final-year students at any of the university veterinary schools in the United Kingdom and Eire.

The closing date for applications is July 31st, and further particulars may be obtained from the General Secretary of the B.V.A.

TOM, DICK OR HARRY?

An American contemporary writes: "Writers are pretty nice people when you get to know them. They are well educated and intelligent . . . . Get your relationship on a first-name basis; don't ignore them and never fight them."

Tuberculosis in Goats caused by the Avian Type Tubercle Bacillus

BY

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AND

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SUMMARY.—An outbreak of tuberculosis in a herd of goats, caused by the avian type tubercle bacillus, is described. The organism was recovered from 3 goats on post-mortem examination and typing was carried out by cultural and biological tests. One of the 3 goats was never tuberculin tested but 10 out of 85 animals, including the other 2 slaughtered for examination, gave avian reactions to the tuberculin test. The origin of the infection is thought to have been wild birds.

NATURALLY acquired tuberculosis in goats is rare. Griffith (1928) considered that the infrequency of the disease was due to factors of environment rather than constitutional resistance, and the literature has been reviewed recently by Francis (1958) and Luke (1958). In the majority of cases of tuberculosis in goats in which the type of the infecting organism has been studied, the disease was caused by tubercle bacilli of the bovine type, and only a few cases in which goats have been naturally infected with avian type tubercle bacilli have been recorded.

Van Es and Schalk (1914) mentioned 2 cases which had been described by Steriopulo, and according to Plum (1935) Wwendensky reported another 3. In Great Britain Griffith (1936) described avian type tuberculosis in the mesenteric lymph nodes of a goat which had reacted to the tuberculin test.

The present paper reports spontaneous tuberculosis in a herd of goats shown to be caused by *Mycobacterium tuberculosis*, avian type.

History and *Post-mortem* Findings

The herd comprised approximately 70 home-bred Saanen goats kept as a supply of experimental animals. There was no contact with other farm animals or domestic poultry although the goats ran in the open on pastures for part of their lives. Goats were occasionally purchased from a dealer but these were kept in isolation in individual pens or in a yard and were never run with the home-bred animals.

Periodic tuberculin tests were carried out on the herd. The last test was 2 years before the first case of tuberculosis was found; at that test all the goats were tested intradermally with johnin and with avian and mammalian tuberculins, and at 72 hours most animals showed increases in skin fold thickness of less than 2 mm. Two adult females had increases of 3 and 4 mm. to avian tuberculin only and 1 adult female 3 mm. to all 3 injections.

Case No. 1. The subject was a 2-year-old home-bred female Saanen, No. 41, which died suddenly after an attack of acute enteritis. The *post-mortem* findings confirmed a diagnosis of enterotoxaemia but some mesenteric lymph nodes were enlarged and contained caseous and calcifying lesions resembling tuberculosis. There was no evidence of similar infection elsewhere in the carcase. The general condition of the animal up to the time of death was good. This goat was never tuberculin tested as it had been born since the previous test of the herd.

At a tuberculin test of the herd carried out immediately following the autopsy findings on goat No. 41, all 85 home-bred and purchased goats were completely negative to avian and mammalian tuberculins and johnin except for the following:—

No. of animal	Reaction to avian tuberculin	Reaction to mammalian tuberculin	Reaction to johnin
A4	12 mm.	6 mm.	2 mm.
17	15	16.5	21
23	11.5	10	16
9	12	1	15
71	10.5	5	11.5
24	9	1.5	4
21	5.5	1.5	9
29	4	0	7
11	3.5	0.5	0.5
39	3.5	0	0.5

Apart from No. A4 which was a purchased cross breed, all reactors were in 1 group of castrated male animals running at pasture. There had been no contact between goats 41 and A4 both of which had also been kept separately from the castrated male reactors.

Case No. 2. Goat No. A4, an adult female cross breed, was purchased 5 months previously from a dealer. This animal was killed immediately following the tuberculin test and a *post-mortem* examination was carried out. The condition of the carcase was good. The bronchial, mediastinal, hepatic, mesenteric and ileocaecal lymph nodes were all enlarged and contained caseous and calcifying lesions. There were several pale, fibrous nodules on the surface of the liver and a single pale subcapsular nodule in the spleen.

Case No. 3. The remaining reactor animals were in the isolated group of home-bred castrated male goats which were running out on pasture all the time. These animals were all in very good condition with no signs of disease. Goat No. 17 in this group

had given marked reactions to both avian and mammalian tuberculins and to johnin and it was decided to kill it for examination. The carcase was in good condition and well covered with fat. Pale nodular areas were apparent in the retropharyngeal and mediastinal lymph nodes but no caseation was seen. Petechial haemorrhages were present in many of the mesenteric lymph nodes and pale greyish caseous nodules up to 5 mm. in diameter were found in 2 of these lymph nodes, mainly confined to the cortex. No abnormality was found elsewhere in the carcase.

Laboratory examination was carried out on material from all 3 goats examined *post-mortem*.

Laboratory Tests

Acid-fast bacilli were seen in films of the lesions from all 3 goats and, although rather pleomorphic, they could not be differentiated by their morphology from tubercle bacilli of the bovine type.

Material from the lesions was treated with oxalic acid and inoculated intramuscularly in each case into 2 guinea-pigs. Single intradermal comparative tuberculin tests were carried out on all the guinea-pigs at 3 and at 4 weeks after inoculation. No reactions were obtained to either avian or mammalian tuberculin at 3 weeks but at 4 weeks all animals gave specific avian reactions. The guinea-pigs were examined at necropsy at either 4 or 6 weeks after inoculation and no lesions were found.

The treated material from the goat lesions was also inoculated in each case on to 10 tubes of a modification of Stonebrink culture medium (Lesslie, 1959). Colonies of acid-fast bacilli were visible in all tubes within 14 days. Growth from 21 days onwards was profuse with over 100 colonies growing in every tube, and was characteristic of the avian tubercle bacillus.

The virulence of the 3 strains recovered on primary culture was tested by further animal inoculations. In each case 2 guinea-pigs were inoculated intramuscularly, one with 1 mg. and the other with 0.1 mg. of moist culture, and 2 rabbits and 2 chickens were inoculated intravenously, 1 of each with 1 mg. and 1 with 0.01 mg. Four weeks after inoculation, tuberculin tests were carried out on the guinea-pigs and chickens and all showed specific avian sensitivity. All guinea-pigs were killed 6 weeks after inoculation and had caseous lesions in the inguinal and lumbar lymph nodes; no other lesions were found. The rabbits inoculated with 1 mg. of culture died within 16 days with the Yersin or septicaemic type of tuberculous infection: no focal lesions were discernible macroscopically but the livers were enlarged and pale throughout and the spleens slightly enlarged and mottled. Numerous acid-fast bacilli were present in the livers and spleens and smaller numbers were seen in films of the kidneys. Of the 3 rabbits inoculated with 0.01 mg. of culture, 1 died at 24 days and the remaining 2 were killed 10 weeks after inoculation, and generalised lesions of tuberculosis were found in all at *post-mortem* examination. The chickens all died between 31 and 41 days after inoculation, with generalised tuberculosis; no significant difference in the results was observed from the 2 different dose rates.

Avian type growth was also obtained on culture media from lesions found in the guinea-pigs, rabbits and chickens.

Material from each of the 3 goats was fixed in 10 per cent. neutral buffered formalin solution and sections were stained with haematoxylin and eosin and by the method of Ziehl-Neelsen. In case No. 1 the lesions in the mesenteric lymph nodes consisted of areas of epithelioid infiltration and giant cell formation with extensive caseation and some centres of calcification. There were streaks of fibrous tissue around the periphery of the lesions. A few acid-fast bacilli were seen. The lesions in the mesenteric lymph nodes of case No. 2 were like those of the previous case and similar changes were also seen in the iliocaecal, hepatic and mediastinal lymph nodes. The liver and spleen showed small areas of epithelioid cell proliferation and giant cell formation but no acid-fast bacilli were seen in these organs. In case No. 3 the retropharyngeal lymph nodes showed epithelioid cell infiltration and in the mediastinal and mesenteric nodes there were areas of caseation necrosis in addition to epithelioid and giant cells.

Discussion

As a result of the *post-mortem* findings in goat 17 (case No. 3), there appears to be little doubt that the other reactors in the same group were similarly infected. However, in view of the nature of the disease and the fact that the remaining reactor animals were in one isolated group, the risk of these goats spreading infection on the premises was considered to be slight and it was decided not to dispose of them all immediately but to keep them isolated and kill them off gradually as replacements became available.

Support for this point of view was provided by the results of a tuberculin test carried out 9 months later. Reactions to mammalian tuberculin and to johnin were much less, and 8 young castrated male goats, run with the infected animals for 6 months, had not become reactors. Furthermore, no lesions were found in 29 test-negative animals (including the daughter of A4) which were killed for experimental purposes.

The presence of well established lesions in the mesenteric lymph nodes only of the goats in 2 of the cases described indicates that the infection was probably acquired by ingestion. The lesions in goat A4 (case No. 2) were more generalised although the animal was in good general health when killed. This was the only infected goat, indicated by tuberculin tests as well as by *post-mortem* findings, which was not home-bred, but the possibility of infection having been introduced into the herd by this purchased animal seems unlikely since goat A4, together with other purchased goats, had been kept in strict isolation in a yard and not mixed with the home-bred animals nor run on the pastures.

The circumstances suggest a natural avian type

infection from wild birds. Large numbers of sparrows and jackdaws were known to feed from the troughs in the fields and even in the goat houses. It also seems significant that most of the reactors to the tuberculin test were in the only group of animals running out on pasture continuously and thus most exposed to this possible source of infection. No definite evidence is available on the existence or otherwise of tuberculosis in the wild birds on this farm but the disease has been reported in jackdaws in this country (McDiarmid, 1956), and in Denmark Plum (1942) found infection in 13.6 per cent. of sparrows on 8 farms where avian type tuberculosis was present in pigs.

The susceptibility of goats to infection with avian tubercle bacilli was investigated by the Royal Commission on Tuberculosis in this country and by other workers. Griffith (1911) found that after exposure of 4 goats by ingestion, tuberculous lesions developed in the lymph nodes along the alimentary canal. In 2 kids which were only 5 days old when fed with the culture, severe ulcerations occurred in the intestinal mucosa and in addition to the lesions in the mesenteric lymph nodes there was tuberculosis of the tonsils, and of the submaxillary, retropharyngeal and portal lymph nodes. All 4 animals were in good condition at time of slaughter. Since goats are susceptible to experimental infection with avian type, it seems likely that a certain amount of naturally acquired infection occurs. The reason that so few cases have been reported may be that few autopsies are performed on goats, and when tuberculosis is found few of the infections are typed.

No clinical signs of tuberculosis were present in any of the goats in the herd involved in this investigation and it appears that infection with the avian type bacillus is not of great importance as a cause of progressive disease in goats. Nevertheless, it may be possible for goats to transmit infection to other domestic animals such as poultry, pigs and cattle by the excretion of avian tubercle bacilli in the faeces. Also, as shown by the work of Griffith (1927, 1931 and 1938) on experimental infection in goats, there is the possibility of avian bacilli being excreted in the milk.

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Clinical Note

"Chronic" Copper Poisoning of Sheep

M. Berwyn-Jones

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SUMMARY.—An outbreak of "chronic" copper poisoning in sheep due to the feeding of a meal rich in copper and primarily intended for feeding to a herd of sows on the same farm is described. Thirty-seven ewes were lost from a flock of 78.

History

FROM December 1st, 1958, a flock of 78 in-lamb ewes were fed on a ration compounded on the farm; this was primarily intended for the feeding of 60 sows and was their normal ration. The ewes were fed at the rate of ½ lb. per head per day until December 25th when this was increased to 1 lb. for about 14 days; thereafter the meal was fed *ad lib.* (*i.e.* up to 3 lb. daily approx.).

This ration had the following composition:—

bran ...	1 cwt.	wheat ...	2½ cwt.
flaked maize	½ cwt.	middlings ...	2½ cwt.
fish meal ...	1 cwt.	mineral mixture	60 lb.
groundnut meal	1½ cwt.	barley to make	1 ton

The copper content of the mineral mixture was 1.75 per cent.

Clinical and *Post-mortem* Findings

The first illness was reported on January 31st, 1959. Examination revealed a staggering, weak ewe with normal temperature, marked jaundice, dull reflexes and emaciated condition: anorexia was noticed in this ewe alone for the first time on that day, and superficial examination of the rest of the flock showed them to be behaving normally. Antibiotic treatment was ineffective and on February 2nd this ewe and one other were found dead.

Post-mortem examination showed generalised jaundice of all organs and tissues, a friable fatty liver with a few liver fluke present, engorged "blackcurrant jelly" kidneys, and dark brown urine in the bladder. There were also some epicardial haemorrhages.

On February 3rd the rest of the flock were treated with carbon tetrachloride; one more death occurred on February 4th.

On February 16th there were 2 more deaths and samples of viscera were sent to Cambridge Veterinary Investigation Centre. The report listed slight fluke infestation, cirrhosis of liver and fatty degeneration, also very congested and enlarged kidneys; there was no evidence of other disease.

Finally, a tentative diagnosis of copper poisoning was made on the evidence of the analysis of the meal combined with the symptoms. Liver samples from 4 animals and a blood sample taken from 1 shortly before death were sent to the Central Veterinary Laboratory, Weybridge, for copper estimations. The liver copper values on a dry matter basis were:—1,100, 1,000, 2,100 and 1,600 p.p.m.; the copper value on a blood sample taken from the last animal

shortly before death was 0.55 mg. per 100 ml.

These values, taken in conjunction with the history and *post-mortem* findings confirmed the diagnosis of copper poisoning.

The meal was not fed after March 4th; although 37 ewes were lost, and 5 ewes aborted before dying.

Discussion

These ewes probably consumed up to 1 g. of copper per head per day. Death in all cases occurred within a few days, and sometimes within 24 hours, of noticeable symptoms.

It is worth mentioning that the sows fed on this ration were unaffected and produced good litters, but it is well known that sheep are very susceptible to copper poisoning (Boughton & Hardy, 1934). Toxaemic jaundice in sheep (chronic copper poisoning) has been intensively investigated in Australia (Albiston *et al.*, 1940; Marston, 1952); there appears to be no harmful effect on the animal until the copper concentration in the liver has reached certain high levels, and further accumulation may lead to sudden breakdown in liver copper storage, resulting in a haemolytic crisis, jaundice and death. Bull (1951) has suggested that various stress factors may precipitate this crisis and in this instance advanced pregnancy may have been the factor; it should be remembered, however, that the remainder of the flock continued to thrive and lambed successfully.

Several outbreaks of chronic copper poisoning in sheep have recently been reported in this country associated with the feeding of copper supplements as well as, or mixed with, a concentrate ration. Clegg (1956) described it in grazing rams given a copperised mineral supplement in addition to 2 to 5 lb. of a concentrate mixture per head per day, while Pearson (1956) reported an outbreak related to the feeding of a concentrate mixture containing approximately 0.07 per cent. of copper sulphate; others have occurred more recently.

Bull (1951) has reported that in chronic copper poisoning, the liver copper concentration is usually higher than 1,000 p.p.m., but an abnormally high liver value alone can not be taken as proof of copper poisoning unless haemoglobinuria is also present, associated with central necrosis of the liver lobules and with a premonitory rise in blood copper. It is interesting that Sutter *et al.* (1958) have reported that high blood copper levels in sheep may be used for a definitive diagnosis of copper poisoning, and that the haemolytic syndrome commonly associated with it is not an essential criterion for its diagnosis. This claim is based on the experimental feeding of 250 mg. copper daily to four 8-month-old sheep. Death occurred after about 10 weeks in 3 of them and after about 14 weeks in the 4th. After 6 to 7 weeks blood serum copper levels were 150 µg. per 100 ml. (0.15 mg. per 100 ml.) and shortly before death were 200 µg. per 100 ml. (0.20 mg. per 100 ml.). The 1 animal which died after about 14 weeks showed no haemolytic signs and no jaundice; its blood serum value was 200 µg. per 100 ml. (0.20 mg. per ml.) and its liver value was 2,160 p.p.m. D.M. This liver value is of the same order as that shown by one of the ewes in the outbreak now reported, but the blood copper value is less than half the pre-mortal rise of 0.55 mg. per 100 ml. found in the ewe whose liver copper value was 1,600 p.p.m. D.M.

Pierson and Aanes (1958) report the treatment of chronic copper poisoning in sheep with molybdenum mixed with sodium sulphate, by adding 50 to 100 mg. of the former with 5 to 15 g. of the latter in water per head, and spraying on hay daily for 3 weeks.

Acknowledgments.—I would like to thank A. Roddis, M.R.C.V.S., for his enthusiasm and assistance in this investigation; also the staff of the Veterinary Investigation Centre, Cambridge, and the Biochemical Dept. of the Veterinary Laboratory, Weybridge, for the copper estimations.

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"THE GRASS ROOTS OF PUBLIC RELATIONS"

Mr. Myron S. Arlein, President, wrote recently in the bulletin of the A.V.M.A. Public Relations Department: "The need for an aggressive, well planned and effective public relations programme for the veterinary profession has been well established by the veterinarian and his organisations. Subsequent to this determination, the solution would appear to be the employment of highly skilled technicians in the art of persuasion and the provision of generous budget to assure a powerfully beamed message. This 'sell' in the vernacular of public relations can only be part of a master plan.

"The major function of our programme must still resolve itself to the aggregate effect of the professional and personal conduct of each veterinarian on his community. Mr. Public in the final analysis will recognise our profession by his personal impressions from actual contact with the veterinarian, his hospital facilities and his service. We must bear the bulk of the responsibility in any projection of public persuasion. There is a definite need for the Madison Avenue artisan in our programme but at the same time each of us should take objective inventory of our professional deportment so that our spokesman's presentation to the public can be integrated with realities."

B.V.A. Annual Congress: Glasgow 1960

WITH the arrival of the New Year the minds of readers will be inclined to turn to summer holidays, among other things, and they may care to think about the Congress to be held in Glasgow from August 28th to September 3rd. The Provisional Committee strongly urges intending delegates to make hotel reservations as soon as possible because the city, a focal point for commerce throughout the year, also becomes an important tourist centre during summer. A preliminary list of hotels has been compiled together with some details of the tariffs, although these may be subject to alteration by August. A fuller list of hotel and hostel accommodation will be published in the early spring. Most of the rooms available in the University will be for men, it should be noted. Those who make reservations from the current list are asked to mention the British Veterinary Association when booking.

Glasgow is relatively near a number of coastal resorts on the Firth of Clyde, themselves popular for holidays and sometimes points of embarkation for the steamers plying among the lochs and islands of the nearer West Coast. While one day of Congress week is to be occupied in sailing, some readers or their families may wish to take further advantage of the facilities for travelling on the Clyde. For them, Helensburgh, Gourock or Wemyss Bay are places to be considered. Glasgow is also conveniently sited for those who wish to take in Congress on their way to or from the Western Highlands and the Islands, such as Arran and Skye. Delegates of a cultural bent may find it worth noting that Congress occupies the second week only of the three-week period during which the Edinburgh International Festival will take place.

Further information on the amenities of Glasgow or its environs may be obtained from offices of the various Town Clerks, or from the honorary local secretary, B.V.A. Congress, 83, Buccleuch Street, Glasgow, C.3.

GLASGOW HOTELS

Hotel and address	Telephone	Number of rooms currently available		Terms B. and B., from	Distance from University (miles)	
		Double	Single			
Central Hotels						
Central Hotel, Gordon Street (Headquarters hotel)	Douglas 9680	20	20	53/–	2	L
North British Hotel, Glasgow ...	Douglas 6711–16	47	36	44/–	2	L
George Hotel, Buchanan Street, Glasgow, C.1	Douglas 6622	40	44	35/6	2	C L
Bath Hotel, 152, Bath Street, Glasgow, C.2	Douglas 7416–9	11	22	30/–	1½	C L
Ivanhoe, 185, Buchanan Street, Glasgow, C.2	Douglas 7284	20	5	33/–	2	L
More's Hotel, India Street, Glasgow, C.2	Central 2641	10	5	39/6	1	C L
Western Hotels						
Botanic Gardens Hotel, Grosvenor Terrace, Gt. Western Rd., Glasgow	Western 2888	8	4	21/–	1	U
Belhaven Hotel, Belhaven Terrace, Gt. Western Road, Glasgow, W.2	Western 7307	10	5	30/–	1	U C
Woodlands Gate Hotel, 11, Lyndoch Place, Glasgow, C.3	Douglas 6123	8	—	21/–	1	U C
Peripheral Hotels						
Boulevard Hotel, Great Western Road, Clydebank	Duntocher 2381	4	4	35/–	5	L
Buchanan Arms Hotel, Drymen, Stirlingshire (for Loch Lomond and the Trossachs)	Drymen 310 & 270	8	8	35/–	20	L

C = Some reduction for children has been specified. L = Licensed. U = Unlicensed.

Current Literature

BOOK REVIEWS

Neutralizing Antibodies in Sera of Cattle Vaccinated against Foot-and-mouth Disease. J. G. VAN BEKKUM. Staatsdrukkerijen Untgeverijbedrigf, 18, Fluwelen Burgwal, The Hague. 175 pages. Price D.f.e. 7-75.

This monograph consists of 2 main sections. The first, and larger, describes a test for the assay of foot-and-mouth disease neutralising antibody and also gives the results of extensive investigations into the variable factors involved in this test. In addition it includes reviews of the literature relating to the antibodies in foot-and-mouth disease and the basic principles of serum-virus neutralisation. The second section deals with observations on the development of immunity in adult cattle and calves vaccinated with typical inactivated vaccines as used in the Netherlands.

The test used to measure neutralising antibody concentration is a modification of that described by Brooksby and Wardle in 1953. Serum-virus mixtures held for 1 hour at 25° C. are added to fragments of surviving bovine tongue epithelium, suspended in a fluid medium in the depressions of a Perspex plate. After a suitable incubation period (3 days) unneutralised virus has multiplied sufficiently to be detected by a complement fixation test. As a routine all sera are inactivated and tested at a dilution of 1 : 10. Each serum is tested against different virus concentrations and the serum titre is expressed as the logarithm of the number of I.D.50 of virus neutralised. Experiments made to help in standardising the technique are described in detail. The results of virus titrations using the tissues from different tongues have been submitted to an analysis of variance. From this it appears that variation in susceptibility of different tongue tissues to the action of virus is considerable in different tests. This effect is most noticeable following the annual field vaccination of cattle with foot-and-mouth disease vaccine. Variation between tests is controlled by the inclusion in each experiment of a reference immune serum.

Due to a variable amount of non-specific neutralisation by the serum of susceptible non-vaccinated cattle, control titrations of the virus suspensions used in each test are made in the presence of an inert diluent and not in normal serum. To minimise the variation between the virus suspensions used in these tests a pool of virus is prepared, sufficiently large to cover a series of experiments, and this is stored at -30° C.

Different neutralisation indices are observed when the same serum is tested under varying conditions of temperature and length of incubation period. The author finds optimum results are obtained when the serum-virus mixtures are incubated for 1 hour at 25° C.

The complement-fixation reaction used as the indicator in this test has also been examined in some detail. The optimum concentrations of hyper-immune guinea-pig serum and the cattle serum under test have been determined and also the rate at which haemolysis progresses with different concentrations of antigen.

It has been shown that dilution of the serum under test results in a decrease in the amount of virus neutralised. The line showing the relationship of the log. of the serum dilution to the log. I.D.50 of virus neutralised is straight and has a regression coefficient of about 1·4.

The author has used this test as a means of studying the rate of development and concentration of neutralising antibody following immunisation with typical inactivated vaccine. The sera of adult cattle showed the presence of antibody one week after primary vaccination. Peak titres were reached in from 2 to 5 weeks and values observed varied from 2·0 to 5·0. After 18 weeks they ranged from 0·8 to 3·2. Considerable differences in the rate of disappearance of antibody from the serum were noted in all experiments. Sera from non-vaccinated cattle showed varying degrees of non-specific neutralisation, but titres greater than 0·5 were observed only occasionally.

Revaccination was shown to be followed by a typical secondary response. Antibody titres considerably greater than those observed following primary vaccination were shown after 7 days and also persisted longer than after primary vaccination. The effect of decreasing the proportion of aluminium hydroxide in the vaccine was investigated and was found to lower antibody titres.

The author has investigated the immune status of calves born of vaccinated mothers. He has shown that they are born with negligible antibody, and that they subsequently receive it from the immune mother via the colostrum. Depending upon the titre of the dam's serum, antibody could still be demonstrated in calves 2 to 6 months of age. Of considerable importance is the observation that traces of maternal antibody can partially or completely suppress the immune response in a young vaccinated animal. A second injection of vaccine 10 weeks after the first provoked only a limited response in some cases. In comparison 6- to 10-week-old calves from susceptible mothers responded normally to vaccination. The author suggests that in the annual vaccination of young stock in the Netherlands, a better result would be obtained if the first dose of vaccine was not given until 6 to 7 months of age, rather than at 2 months as at present.

In 1955–6, some 200 samples of serum from cattle immunised in the field with trivalent vaccine, were examined for type O antibody titres. These samples were taken at different intervals after vaccination and a large percentage of them were of high titre. From the facts that there was a low incidence of foot-and-mouth disease prior to sampling, and that co-incidentally high titres to type A and type C were also shown, the author concludes that the persisting high antibody titres were due to the vaccine and not to secondary stimulation from inapparent infection.

The relationship between antibody titre and resistance to challenge was also investigated. The method of challenge in this instance was the swabbing of the mouth with a cloth drenched with a suspension

of virulent virus. In a series of 122 cattle challenged by this method, 28 had antibody titres of less than 1·2 and were fully susceptible, *i.e.* they showed both primary lesions in the mouth and secondary lesions on the feet. In the histogram illustrating this series only 10 of these susceptible animals are shown, which might on casual examination give a more favourable impression than was intended. In the remaining 95 animals shown in this histogram, antibody titres ranging from 1·2 to 4·2 were observed. Of 45 cattle with sera in the range 1·2 to 2·5, some 25 were completely protected, 19 showed primary lesions only and 2 animals developed primary and secondary lesions. The other 49 cattle in this series had antibody titres of 2·5 to 4·2. All of these animals were completely protected, except one with a titre of 2·7 which developed a localised primary lesion on the tongue.

A less clear relationship was observed between antibody titre and clinical response when vaccinated cattle were challenged by intradermal inoculation of the tongue. Results are given of experiments in which groups of cattle were challenged with 1,000 I.D.50 of virus and also from cattle challenged with 10,000 I.D.50. In a group of 55 cattle challenged with 1,000 I.D.50, 31 had antibody titres varying from 0·7 to 2·5. Of these 6 were completely protected, 22 had primary lesions at the site of tongue inoculation and 3 developed both primary and secondary lesions. Paradoxically the animal with the titre of 0·7 showed a lesion on only 1 foot whilst the other 2 animals with titres of 2·2 and 2·3 were fully susceptible, showing complete generalisation. In a group of 19 cattle challenged with 10,000 I.D.50 antibody titres ranged from 0·9 to 4·5. All of these animals showed primary erosions. Of 7 cattle with titres between 0·9 to 3·2, 4 developed foot lesions, and 2 of the remaining 12 in the range 3·3 to 4·5 also showed generalisation.

Protection against the development of primary lesions at the site of inoculation of the tongue was shown to be related to the antibody titre observed, but if a large enough dose of virus was inoculated, a vesicle could always be produced.

This work will be of considerable interest to those engaged in research on immunity to foot-and-mouth disease. It is published in Dutch, the figures having captions in Dutch and English. There are also short summaries in English, German and French.

Pendley and a Pack of Hounds. By DORIAN WILLIAMS. Hodder & Stoughton Ltd., London. (1959). 21s.

Mr. Dorian Williams has interests ranging from Shakespeare to fox-hunting, and from adult education to television. All these, together with a lifelong love of horses and a fixed determination to preserve his family home, Pendley Manor, have enabled him to write an unusual and interesting book.

The story of the author's success in turning Pendley into a centre for adult education, despite post-war shortages of staff and equipment and recurring financial crises, is an inspiring example of individual achievement. But although there is an underlying seriousness of purpose, he never fails to see the humorous side of the awkward situations which accompany this new venture. Also, he describes most entertainingly a number of colourful personalities amongst his staff and students.

After 5 years' hard work, during which time he owned no horses, an unexpected day's hunting aroused all his former enthusiasm for the sport, which he took up again with tremendous enjoyment. Later, in addition to his directorship of Pendley, he became a Master, first of the Grafton Hunt and subsequently of the Whaddon Chase. All horse-lovers will enjoy this second half of the book with its descriptions of horses, riders and hunting.

Many excellent illustrations show scenes varying from Shakespeare's plays produced in Pendley gardens, to pictures of the author's horses and the Whaddon Chase hounds.

Brief Course in Organic Chemistry. Behr, L. C., Fuson, R. C., Snyder, H. R. (1959). 2nd ed. John Wiley and Sons, Inc., New York (Chapman & Hall, London). 46s.

There must be a very large number of scientific workers who have to use some of the many aspects of chemistry, but feel considerable regret that it is virtually impossible to keep abreast of modern developments in this rapidly expanding field. The authors of this book, all of whom are well known American chemists, have attempted to alleviate the problem by producing the second edition of this relatively short book (289 pages).

This type of book, where much is condensed into a little space—only a few hundreds of the 2,000,000 known organic chemicals are mentioned—is apt to be criticised for its omissions, but there is no pleasing everybody.

The subject matter is clearly treated in a more or less classical style, each chapter ends with a few questions, and the general production is excellent. It is very pleasing to find that there are many references to such well known substances such as detergents, antibiotics and plastics. The biochemical comments are valuable to the non-biological chemist, as are the details of manufacture to the biochemist. Each chapter starts with a diagrammatic structural formula of the group prototype, but the reviewer, albeit a little old-fashioned, prefers the more "open" type of structural model where the separate atoms can be seen more clearly than in the modern "close" type of model.

The illustrations of the polysaccharides are not very good, and the wavy and tendril-like valency links are a little misleading in appreciating the structure. Likewise it is noted that no attempt has been made to represent the sterols by modern configurational methods, even though a whole chapter has been devoted to the classical aspects of optical isomerism. The chapter on the alkaloids is good, and a very reasonable selection of structures has been presented, although it is perhaps a pity that lysergic acid has been omitted, particularly as its synthesis has been reported by Woodward and his school some time ago.

In spite of these criticisms, this is a useful book, and should be included on the shelves of the reference library, if not in the private collection.

Profitable Pig Farming by Geoffrey Johnson (1959). Dairy Farmer (Books) Ltd. Ipswich. 25s.

Success stories are common enough. There is always a little romance about them and albeit at times a little envy. Some are just a matter of luck, others the result of careful observation and practice over the years. Mr. Johnson's is of the latter type and as such deserves serious consideration.

All aspects of pig production are dealt with including the general organisation and the economics of the whole system, building construction, breeding and disease. Sometimes, as may be expected, the author's interpretations will not be generally accepted but most of his views, welded in the fire of necessity, are based upon extensive experience.

Points from his programme are as follows:—The key to efficiency is a clockwork routine of services and farrowing, a process which in itself puts great demands on the management. The value of feeding the sow well, from a month just before service to just a month afterwards to ensure adequate "setting" of the fertilised ova will be generally accepted, but dependence of the breeding stock on grass and/or sunshine for vitamins A and D comes as a surprising suggestion from one so experienced. In a country where the latter is scarce and pastures are mostly of debatable quality, premixes of stabilised vitamins are surely to be recommended. Furthermore, who will agree that oedema disease is common in association with a high percentage of oats in the ration? If this is so what is the cause of the other 99 per cent. of cases that give national concern? The benefits of hybrid vigour are dismissed with some disdain which is surprising in view of the amount of evidence to support this trend. It is interesting to note that Mr. Johnson considers injection of very young pigs a difficult process. This attitude in an experienced person is highly significant. So difficult does he find this in fact that he is prepared to adopt the more tedious process of daily dosing with standard copper/iron mixtures, or sprinkling iron sulphate on a clean patch of floor.

Veterinarians in particular will be interested in the recommendation for the building up of virus-free herds, but will be less enthusiastic over the use of an ointment made of oil of tar and linseed with sulphur for the treatment of mange.

One may not wish to follow the author's example in every detail especially when he lapses into antiquity, but his experiences provide a valuable basis to the complex problem of pig husbandry and for this reason alone are well worth serious reflection.

The Calf : Its Management, Feeding and Health by J. H. B. Roy, M.A., DIP.AGRIC., PH.D. (1959). Farmer and Stockbreeder Publications Ltd. London. Price 9s. 6d.

To call a book a handbook is merely to encourage the temptation to put it back on the shelf for future reference when time allows and when one is in a more relaxed frame of mind.

This second edition is perhaps better described as a "handybook" for quick reference.

No practical aspects of the rearing and management of the calf have been overlooked. Much new information has been added, it is well illustrated and has many useful tables. These cover such matters as—the average growth rate curve of the various breeds, the weight of the heifer in accordance with its girth measurement, the composition of colostrum. Of particular interest is the weekly feeding schedule for various methods of rearing calves to 6 months.

Such an extremely topical problem as the methods of calf rearing is dealt with in considerable detail both from the point of view of the practical man and the specialist enquiring into the differing food requirements of the animal at various stages of growth.

Information on dehorning, castration or dosing for worms is given in full and there is a section on disease which is well written and up to date including the modern methods of treatment for husk and warble fly. The latter section, however, will be of less interest to veterinarians than those dealing with management and feeding.

R.C.V.S. OBITUARY

We record with regret the deaths of the following members of the profession:—

KEPPEL, John Joseph Guiney, O.B.E., Spier Lyndoch, Stellenbosch, South Africa, Major late R.A.V.C. (M.). Graduated December 14th, 1911, at the Veterinary College of Ireland, Dublin. Died October 1st, 1959, aged 75 years.

The following obituary notice of Major Keppel appears in the Journal of the South African Veterinary Medical Association:—

Major Keppel was born in 1884 in County Cork. He was a keen sportsman and played rugby for Ireland from 1905 to 1912. He was also an oarsman and polo player.

He came to South Africa in 1912. During World War I he served with the Royal Army Veterinary Corps and with the 12th Lancers. He was awarded the O.B.E. and was four times mentioned in despatches. He was stationed in many parts of the Union, including the Transkei, Potchefstroom, Middelburg (Tvl), Piet Retief, Dundee, and as Sub-Director of Veterinary Services at Bloemfontein and Cape Town.

He retired in 1944 and a year later purchased the old historic farm of Spier Lyndoch near Stellenbosch. Here, with his son, he farmed wine, tobacco and Jersey cattle.

He leaves a wife, a son and a daughter. To them we extend our heartfelt sympathy.

PARSONS, Charles Colin, of Exeter Street, Launceston, Cornwall. Graduated London, July 14th, 1905; died December 23rd, 1959, aged 76 years.

WILSON, Thomas, Barker Street, Nantwich, Cheshire. Graduated August 25th, 1907, at the Royal (Dick) Veterinary College, Edinburgh. Died November 23rd, 1959.

Supplementary Veterinary Register

FENN, Harry Victor, 21, Highfield Avenue, Dovercourt, Essex, D.V.M. (ONTARIO), Captain late R.A.V.C. Died November 29th, 1959, aged 89 years.

News and Comment

THE NEW YEAR HONOURS LIST

The following members of the profession and others associated with it have been honoured by Her Majesty in the New Year Honours List:—

C.B.E., Mr. J. B. Randall, Director of Veterinary Services and Animal Industry, Uganda. *O.B.E.*, Mr. S. V. Collard, Ministry of Agriculture, Divisional Veterinary Officer, and Chairman of the Provisional Committee, B.V.A., Congress at Leamington Spa, 1955-6; Mr. A. Orhan, C.V.O., Cyprus; Mr. J. M. Angell, Chairman of the National Pig Progeny Testing Board; Dr. Joseph Edwards, Scientific Adviser to the Milk Marketing Board; Mr. A. T. R. Mattick, lately Departmental Chief Scientific Officer, National Institute for Research in Dairying, Shinfield; Mr. W. T. Price, Principal, The Harper Adams Agricultural College, Newport, Staffs.

A Baronetcy was conferred upon Mr. G. R. H. Nugent, M.P. for Guildford since 1950, and past Parliamentary Secretary, Ministry of Agriculture, Fisheries and Food. Dr. John Hammond, the distinguished agricultural scientist, has become a Knight Bachelor.

PERSONAL

Mr. J. F. Gracey has been awarded the Ph.D. in the Faculty of Agriculture, Queen's University, Belfast. His thesis was entitled "A Study of Disease Incidence and Wastage in Livestock in Northern Ireland."

Mr. T. D. Lloyd-Jones, veterinary officer, who was stationed at Nantwich, has retired from the service as from December 28th, 1959.

Mr. G. H. Locke. Their many friends in the profession will wish to extend congratulations and good wishes to Mr. and Mrs. Locke, of Oxted, who celebrate to-day (Saturday) the sixtieth anniversary of their wedding. Mr. Locke is a past-President of the R.C.V.S. and an honorary life member of the B.V.A., on whose behalf a telegram of greetings was sent from Headquarters.

Dr. N. H. Pizer has been appointed Senior Advisory Officer (Science) in the National Agricultural Advisory Service in succession to Mr. W. Morley Davies who retired on December 31st, 1959. Mr. Morley Davies at the same time gave up the secretaryship of the Agricultural Improvement Council for England and Wales, which he held since 1949; Miss J. B. E. Haydon and Dr. R. E. Taylor have been appointed joint secretaries.

Births

STIRLING.—On January 3rd, 1960, at Witney, to Aileen, wife of L. R. Stirling, a son, Allen George.

WILLIS.—On December 27th, 1959, at 18, Woodgreen Road, Luton, Beds., to Gillian Willis, B.V.M.S., M.R.C.V.S. (*née* Wickens), wife of John Willis, B.SC., a son, Nicholas Stephen John.

COMING EVENTS

January

12th (Tues.) Meeting of the B.V.A. Technical Development Committee at 7 Mansfield Street, London, W.1., 10.30 a.m.

13th (Wed.) Meeting of the Southern Counties Veterinary Society at the Royal Hotel, Winchester, 7.30 p.m.

Joint Meeting of the Society of Practising Veterinary Surgeons with the Southern Counties Veterinary Society as listed above.

20th (Wed.) 11th Anniversary Dinner-Dance of the University of Bristol Centaur Society at the Berkeley, Clifton, Bristol.

Meeting of the Warwickshire Veterinary Club in the Regent Hotel, Leamington Spa, 8 p.m.

23rd (Sat.) Annual General Meeting of the East Anglian Pathologists' Club at the School of Veterinary Medicine, Madingly Road, Cambridge, 2 p.m.

28th (Thurs.). Meeting of the Western Counties Veterinary Society at the Rougemont Hotel, Exeter, 2.30 p.m.

February

3rd (Wed.) Meeting of the Southern Counties Veterinary Society at the Royal Hotel, Winchester.

5th (Fri.) Annual Ball of the Royal (Dick) School of Veterinary Studies in the Assembly Rooms, George Street, Edinburgh, 9 p.m.

BRITISH VETERINARY ASSOCIATION

Quarterly Meetings in London

Wednesday, January 20th, at 7 Mansfield Street.
10 a.m. Farm Livestock Committee.
11.30 a.m. Parliamentary and Public Relations Committee.
2.15 p.m. Veterinary State Medicine Committee.
4.30 p.m. Home Appointments Committee.

Thursday, January 21st, at 7 Mansfield Street.
10 a.m. Small Animals Committee.
2.15 p.m. General Purposes and Finance Committee.

Friday, January 22nd, at Connaught Rooms, W.C.2.
10.15 a.m. Council Meeting.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Ayr. Wyllieland Farm, Fenwick, Kilmarnock; Chapelton Farm, Maybole; Raith Hill Farm, Coylton (Dec. 29).
Cumbs. Brackenthwaite Farm, Wilton, Egremont; Oak Tree Hall Farm, Castle Carrock, Carlisle (Dec. 29).
Derbys. Riversdale Farm, Holymoorside, Chesterfield (Dec. 29).
Dumfries. Newmains, Kirkbean (Dec. 31).
Fife. Scotts Walls Farm, Crossgates, Cowdenbeath (Jan. 1).
Lanark. West Netherton Farm, East Kilbride, Glasgow (Dec. 29).

Norfolk. Old Hall Farm, Witton, North Walsham, Norfolk; Melbourne House, North Walsham (Dec. 29).
Salop. Sallins Farm, Piclescott, Leebotwood, Shrewsbury (Jan. 4).
Somerset. Norton Farm, Kewstoke, Weston-super-Mare (Jan. 4).
Stirling. Burnfoot Farm, Dumgoyne, Glasgow (Jan. 4).
Sussex. Bramber Farm, North Mundham, Chichester (Jan. 4).
Westmorland. Brow Farm, Dufton, Appleby (Jan. 4).
Worcs. The Shrouds Farm, Oldwood Common, Tenbury Wells (Dec. 29).

Foot-and-Mouth Disease

Herts. Slaughterhouse, Amwell End, Ware; Slaughterhouse, 5, New Street, Watford (Jan. 4).

Fowl Pest

Essex. 94, Brentwood Road, Ingrave, Brentwood (Jan. 1).
Hants. Rorehill Farm, Middle Wallop, Stockbridge (Jan. 3).
Kent. Cherry Bungalow, Little Cossington, Aylesford, Maidstone (Dec. 26); Haylands Farm, Smarden, Ashford (Dec. 28); The Grange Poultry Farm, Kenardington, Ashford (Dec. 30); Mount Castle Farm, Lenham, Maidstone; Coles Farm, Bough Beech, Edenbridge (Dec. 31); Snughorn Farm, Smarden, Ashford (Jan. 1); West Hoy Farm, Smarden, Ashford (Jan. 3).
Norfolk. Upgate Swannington, Norwich; West End Farm, Tibenham, Norwich; Aylsham Road, Swanton Abbot, Norwich; Woodsend, Framlingham Earl, Norwich; Wessex Farm, Thrandeston, Diss; Old Home Farm, Brooke, Norwich; 3, White Hart Street, East Harling, Norwich; Valley Farm, Bintree, Dereham; King Street, East Harling, Norwich; Manor Farm, Larling, Norwich; Leningsede, Larling, Norwich; Mill Lane Farm, Weybread, Diss; The Homestead, Wortham, Diss (Dec. 27); Rectory Farm, Larling, Norwich; King Street, East Harling, Norwich; Honeypatch Blofield Corner, Blofield, Norwich; Long Road, Framlingham Earl, Norwich; Old Rectory, Little Fakenham, Thetford; Cannister Hall, Houghton in the Dale, Walsingham (Dec. 28); Woodside House, Hainford Road, Stratton Strawless, Norwich; Little London Farm, North Walsham; Little London Farm, Corpusty, Norwich; 4, Gallants Lane, East Harling, Norwich; Toft Mead, South Green, East Dereham; Street Farm, Aslacton, Norwich; Devonia, Cross Road, Upper Stoke, Holy Cross, Norwich; Broad Marsh Farm, Great Ellingham, Attleborough; Old Hall Farm, Badley Moor, Dereham; Fir Covert Road, Felthorpe, Norwich (Dec. 29); Westcarr Road, Attleborough; Brackthorn Meadow, Redgrave, Diss; Mill Road, Barnham Broom, Norwich; Modle Farm, Stoke Holy Cross, Norwich; Rose Villa, The Street, Horham, Diss; Ley Farm, Shotesham, Norwich; Wilsons Farm, Swanton Abbot, Norwich; Sunnydene, The Green, Old Buckenham, Attleborough; Mayhurst, Salhouse Road, Rackheath, Norwich (Dec. 30); The Black House, Wortham, Diss; Brackendale, Upper Stoke, Norwich; The Arch Cottage, Westwick, Norwich; Old Hall Farm, Old Buckingham, Attleborough; Rookery Farm, Scottow, Norwich; West Park Farm, Ickburgh, Thetford; Homlea, Cherry Tree Road, Tidenham, Norwich; 4, Railway Cottages, East Harling, Norwich; The Willows Farm, Moulton St. Michael, Norwich (Dec. 31); Glenlodge Farm, Bawburgh, Norwich; Hall Farm, Rackheath, Norwich; The Nurseries, London Road, Attleborough; Boundary Cottage, Upper Stoke Holycross, Norwich; Holme Sheds, Pond Farm, Holme Hale, Thetford; Near Rectory, Swanton Abbot, Norwich (Jan. 1); Chestnut Farm, Hingham, Norwich; 17, The Row, Scottow, Norwich; Long Meadow, The Street, Brundall, Norwich; The Laurels, The Street, Brundall, Norwich; King's Head Inn, Denton, Harleston; 47, Shelfhanger Road, Diss; Station House, Buxton, Norwich (Jan. 2); Grove Farm, Flebrigg Roughton, Norwich; The Flaxlands, Carleton Road, Norwich; Manor Farm, Blofield, Norwich; Glebe Farm, Denton, Harleston; Hedges, High Green, Brooke, Norwich; 10, Hamilton Road, Cromer; Green Farm, Thompson, Thetford; 4, Reepham Road, Briston, Melton Constable (Jan. 3).
Northants. Middle Street, Farcet, Peterborough (Dec. 31); 36, Field Terrace, Farcet, Peterborough (Jan. 3).
Notts. 21, Highill Lane, Linby; Leyfields, Kneesall, Newark (Dec. 26); Orchard Farm, Main Street, Blidworth, Mansfield; Carr Banks Farm, Farnsfield, Newark (Dec. 30).
Suffolk. 1, Kirby Rise, Barham, Ipswich (Dec. 26); Walnut Tree Cottage, Cow Green, Bacton, Stowmarket (Dec. 27); The Mill, Mill Road, Occold, Eye (Dec. 28); 216, Thornham Parva, Eye; Broiler Plant, The Green, Kirby Cane, Bungay (Dec. 29); Willow Farm, Cromford, Snape, Saxmundham; Cranley Hall, Eye (Dec. 30); Grange Farm, Wyverstone, Stowmarket; The Hall, Badwell Ash, Bury St. Edmunds; 12, High Street, Lakenheath, Brandon; Rolls Farm, Helions Bumpstead, Haverhill; Stanwell Lodge, Thorndon, Eye (Dec. 31); Hope Cottage, Brown Street, Old Newton, Stowmarket; The Lodge, Great Ashfield, Bury St. Edmunds; Linton Farm, Beck Row, Bury St. Edmunds; The Limes, Eriswell Road, Lakenheath, Brandon; Cliff Farm, 34, Beck Row, Bury St. Edmunds (Jan. 2); Rectory Road, Bacton, Stowmarket; 29, St. John's Street, Beck Row, Bury St. Edmunds (Jan. 3).
Surrey. Rosemead, Lickfolds Road, Rowledge, Farnham (Dec. 31).
Sussex. Mill Lane Piggery, Maresfield, Uckfield (Dec. 28); The Old Flax Factory, Five Ash Down, Uckfield; 7, Millwood Lane, Maresfield, Uckfield; Woodlands Mill Lane, Maresfield, Uckfield (Jan. 3).
Warwicks. Bidford Grange, Bidford-on-Avon (Jan. 2).
Worcs. New Larkborough Farm, Bretforton, Evesham (Dec. 27).

Swine Fever

Cambs. Cantelup Farm, Haslingfield (Jan. 1).
Ches. Hill Top Farm, Thornton Common Road, Thornton Hough, Wirral (Dec. 29).
Cornwall. Wheal May Farm, Leedstown, Hayle; Rinsey Farm, Aston, Helston (Jan. 4).
Derby. Corner House, Findern (Jan. 1).
Devon. Trenchar, Eggesford, Chulmleigh (Dec. 29).
Dorset. Rosedale Farm, Holt, Wimborne (Dec. 29); Lower Bagber Farm, Bagber, Sturminster Newton; Manor Farm, Little Canford, Wimborne (Dec. 30); Sandpit Mill Farm, Broad Windsor, Beaminster; Binley, Waterloo Road, Corfe Mullen, Wimborne (Jan. 4).
Essex. Piggeries, Daws Heath Road, Rayleigh (Dec. 29); Little Boyton Hall, Roxwell, Chelmsford (Dec. 30); Radley Green Farm, Radley Green, Roxwell, Chelmsford (Jan. 1).
Hunts. High Street, Sawtry (Jan. 1).
Lincs. 13, Sluice Road, Holbeach, Spalding (Dec. 30).
Mon. Red House Farm, Llanarth, Abergavenny (Jan. 4).
Mont. Hirrhas Hall, Llanerfyl; Glyn Farm, Brooks, Berriew (Dec. 29); Hendre Farm, Llandrinio, Llanymynech (Jan. 1); Yew Tree Farm, Llanfair Caereinion, Welshpool (Jan. 4).
Northants. Knuston Home Farm, Irchester, Wellingborough (Dec. 29); Manor Farm, Easton Maudit, Wellingborough (Dec. 30).
Oxford. Lamberts Barn Farm, Cropredy, Banbury (Dec. 29).
Salop. White House Farm, Weston Lullingfields, Shrewsbury (Dec. 30); Lacon Farm, Prees, Whitchurch (Jan. 4).
Somerset. Millslade Farm, Ashcott (Dec. 20); Sadbrook Farm, Hinton Blewett, Temple Cloud, Bristol (Dec. 30).
Suffolk. Hall Farm, Lidgate, Newmarket (Dec. 29); Milbeck Farm, Mildenhall, Bury St. Edmunds (Dec. 31).
Sussex. Lidsey Lodge Farm, Lidsey, Bognor Regis (Dec. 31).
Warwicks. Llamas Farm, Kilsby, Rugby (Dec. 31); Bells Farm, Bells Lane, Birmingham (Jan. 4).
Wilts. The Bungalow Farm, Newton Toney, Salisbury (Dec. 29); Halfway Farm, Beanacre, Melksham (Jan. 4).
Worcs. Bowens Place, Baveney Wood, Neen Savage, Cleobury Mortimer, Kidderminster (Dec. 29); Half Key Farm, Malvern (Jan. 4).
Yorks. 449, Manor Lane, Sheffield, 2 (Jan. 1).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

A Veterinary Television Talk

Sir,—Though fully realising that it is not my business, may I be permitted to occupy a little of your valuable space in response to—*not* replying to—the letter by Mr. D. H. Phillips of Eccleshall, published last week?

I gather, by and large, that Mr. Phillips is vehemently complaining about the fact that a member of our profession has the audacity to appear on television and expound *his own* theories on parturition. What a wicked thing to do; at Christmas time as well! How much better for us all if the broadcaster had expounded Mr. Phillips's views; that is, of course, if Mr. Phillips has any original views—or perhaps like many of us he is still "pulling and hoping."

I would like to suggest seriously to Mr. Phillips and all who think along similar lines, that they approach the B.B.C. T.V. Agriculture Service at Birmingham. They will find the producer of "Farming" a very charming and intelligent man with a degree from one of our senior universities, who will be only too glad to listen to some cut-and-dried programme on parturition. It is no use approaching him with hazy ideas, for the producer is a very busy man. These ideas have got to be crystallised. The mystery television star (!) to whom Mr. Phillips takes exception would be the last person to claim any monopoly on B.B.C. television, but I would seriously remind Mr. Phillips—and here I speak from a little experience—that there is no medium so exacting for those who take part as television.

But go to it Mr. Phillips! Don't waste any more time writing letters, get busy with that script *now*; but remember, your facts must be right when "Big Brother is watching you."

Yours faithfully,
ROGER MASSEY.

Mill House,
Mill Lane,
Stock,
Essex.
January 4th, 1960.

Sir,—I would like to comment on your correspondent's letter which appeared under the above heading in last week's RECORD. Realising the necessity for brevity, I will confine myself to the following points:—

There can be no shadow of doubt that our television veterinary personality ("T.V.P.") has the responsibility of our profession at heart, and I am also certain that in him we are very fortunate in having someone not only forthright and progressive in his views, but with extremely good histrionic ability—in fact someone who would be a credit as a P.R.O. to any profession.

Regarding the usefulness of this talk to the profession I think I am quite in order in saying that probably 80 per cent. of the profession could benefit from it. It might make some of us realise (if realisation has not dawned already) that some of our former methods were archaic in the extreme, to the detriment of our patients.

Regarding your correspondent's view that the broadcaster's arguments were nonsense, may I ask if he is writing from vast experience, or is he too young to respect the experience of a clear-thinking veterinary surgeon, with, if I may be permitted to say it, much vaster experience over a much greater period of time.

Having had myself approximately 18 years in agricultural practice, and having learned the hard way, *i.e.* by trial and error, I can assure your correspondent that I am in full agreement with our broadcaster's views. It is naturally easy to pick out exceptions to any generalisations, but these in the main only serve to confirm the basic principles.

Many people who watched this programme have talked to me about it, and the unanimous verdict of all was that the programme was absolutely first-rate, absorbing, and a great credit to our small profession.

In conclusion, may I state that I feel a trifle jealous that our T.V.P. thought of putting over this idea before I did. I wonder if your correspondent is not suffering from a little of the same complaint.

Yours faithfully,
F. W. LOGUE.

93, Head Street,
Halstead,
Essex.
January 2nd, 1960.

Sir,—I viewed with some interest the programme on calving. Although it was directed mainly to farmers, I am sure the broadcaster will forgive friendly comment from a colleague on a few points.

The film on calving presented within the programme fully justified his remarks on the wonders of nature; but how wrong he is to assume that herd owners and stockmen are incapable of making the decision when to attempt to calve a cow. The first shot showed the veterinary surgeon making a vaginal examination and remarking that the *os uteri* was only half open. Under no circumstances attempt to calve the cow or interfere at this stage, he said. Surely he credits the herd owner and stockman with more sense? I cannot imagine the owner asking his stockman to drive a car out of the garage through a half-open door. Surely both realise that irreparable damage would result?

I fully agree with the speaker on the preparation of a well-bedded loose-box, etc., for calving purposes but do not agree with his reasons why these things are necessary. He remarked that most

injuries at parturition are caused by the animal slipping, but here our opinions differ.

The loose-box idea is based on the psychological fact that an animal will calve much more easily when loose and presented with an environment more akin to the wide-open spaces of nature. Even when labour pains are severe and acute in the bovine animal it will seldom throw itself down without warning, on the floor or loose its footing. Both these traits are common to the equine species but not to the bovine. Careful study of the film presentation shows how the animal eases itself to the ground.

The most frequent injury at calving, in my experience, is a pressure or obturator paralysis caused by continual pressure of the foetus on the obturator nerve as it passes through the obturator foramen on the floor of the pelvis.

This brings me to another point of disagreement: that of waiting 2 to 4 hours when labour commences before making an examination. At the onset of labour nature brings into force many aids, including muscular contractions of the uterus and abdominal muscles, and an increased intra-abdominal pressure. When the foetus reaches full term it becomes a foreign body in the womb and these aids and contractions are purely concerned with the expulsion of that foreign body. To that end we, as veterinary surgeons, should contribute as quickly as possible.

Continuous pressure of the foetus on its journey through the pelvis damages the obturator nerve and brings about this "pressure" paralysis. This severe pressure can be well appreciated if one gets the hand caught between the pelvic rim and the foetus. Injury due to slipping, if it occurs at all, invariably is due to rupture of the round ligament of the femur at its insertion into the acetabulum and is not in my opinion as drastic a setback as paralysis. Even with rupture of this ligament the animal can at least get up and walk on 3 legs; but with pressure paralysis a continuously recumbent state for a long period is very detrimental both to the health and the milking potential of the animal.

The third point on which I would like to comment is posterior presentation, and why, in my opinion, prompt veterinary attention is necessary.

When one is called to such cases the water sac is usually ruptured by the sharp hind feet of the calf, in contrast to anterior presentation where the round nose of the foetus precludes this happening. Uterine fluids are being expelled rapidly by the continuous uterine contractions and the calf is often dead. In my opinion this is due, not as the broadcaster suggests, to the calf taking a deep breath and inhaling uterine fluids when the hind quarters react to the colder air, but by the intrauterine pressure driving the fluids into the mouth of an already dead calf; dead because the placental attachments to the uterine wall have already become dislodged by waiting too long. This is borne out in my experience by the fact that in a high percentage of cases of posterior presentation the placenta comes away in one piece immediately the calf is born.

My advice to farmers is to provide their animals with as near natural conditions as possible. Leave cows and heifers loose in the winter in open courts, bedded with dung and overlying straw simulating the grassy sward, open to the sky or under cover according to their pleasure, and very little trouble will occur at calving time. Domestication and its aftermath have so much reduced the health and general muscle tone of our animals that veterinary surgeons can never be done without. My limit of waiting time at parturition would be one hour and not 2 to 4 as the commentator suggested.

Yours faithfully,
BASIL H. HART.

Levally,
Enniskillen,
N. Ireland.
December 27th, 1959.

Sir,—Many veterinary surgeons in private practice will agree wholeheartedly with much of what Mr. D. H. Phillips says in his letter (THE VETERINARY RECORD, January 2nd, 1960).

It is, of course, undesirable that one man, holding a minority opinion, should be able to put it across so powerfully and apparently so authoritatively, without public contradiction. But an important aspect of this subject is that even if he is absolutely correct in his ideas, the effect of his broadcasts has been bad. "A little knowledge is a dangerous thing," runs the adage. The average farmer cannot follow and remember the whole argument. What he remembers is that the veterinarian on television (who, of course, knows more than you!) seems to advocate waiting, and giving the animal time. He cannot be expected to appreciate all the possible causes of dystocia. The effect of such "knowledge" is not very beneficial either to himself or to his veterinary surgeon.

Yours faithfully,
G. HUDSON.

91, Main Street,
Pembroke,
South Wales.
January 2nd, 1960.

Veterinary Service in Saskatchewan

Sir,—Further to my letter of September 11th, 1959, which was published in the October 3rd issue of your magazine, would you please be good enough to add that information regarding veterinary matters in Saskatchewan can also be obtained from Saskatchewan House, London, as well as from the secretary of the Saskatchewan Veterinary Medical Association.

Inquiries with regard to practice vacancies in this province should be forwarded to Dr. V. E. Senior, Administration Building, Regina, Saskatchewan, who is responsible for the administration of the Veterinary Service District scheme.

Yours faithfully,
W. TURNBULL,
Secretary-Treasurer.
The Veterinary Medical Association of Saskatchewan,
City Hall,
Saskatoon,
Saskatchewan,
Canada.
December 11th, 1959.